The Tachykinins Substance P and Hemokinin-1 Favor the Generation of Human Memory Th17 Cells by Inducing IL-1β, IL-23, and TNF-Like 1A Expression by Monocytes

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Résumé en anglais	The nervous system influences immune responses through the release of neural factors such as neuropeptides. Among them, the tachykinin substance P (SP) signals via the neurokinin 1 receptor (NK-1R), which is expressed by various immune cells. We thereby analyzed in this paper whether tachykinins may participate in human CD4+ Th cell polarization. We report that SP and hemokinin-1 (HK-1) upregulate IL-17A and IFN-γ production by human memory CD4+ T cells without affecting IL-4 and IL-10 production. SP and HK-1 switch non–Th17-committed CD4+ memory T cells into bona fide Th17 cells and Th1/Th17 cells. In contrast, SP and HK-1 do not modulate the polarization of naive CD4+ T cells. SP- and HK-1–induced Th17 cell generation is mediated through NK-1R and requires the presence of monocytes. SP and HK-1 trigger IL-1β, IL-6, and TNF-α production, upregulate IL-23 production, and enhance TNF-like 1A expression on monocyte surface. Neutralization experiments demonstrated that IL-1β, IL-23, and TNF-like 1A are involved in the SP- and HK-1–induced Th17 cell. The other members of the tachykinin family, neurokinins A and B, have no effect on the differentiation of naive and memory T cells. These results thereby show that SP and HK-1 are novel Th17 cell-inducing factors that may act locally on memory T cells to amplify inflammatory responses.
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